Is a treatment for pityriasis rosea really needed?

Sir,

We read with interest the article by Jairath *et al*.[1] about the use of narrowband ultraviolet B (NB-UVB) phototherapy in pityriasis rosea (PR) that prompted us to make some considerations.

First, regarding the pathogenesis of PR, we point out that it is questionable to define "anecdotal" the huge amount of studies available documenting its viral etiology since 1987, studies that have never been mentioned in the paper. Remarkably, these studies have been performed using the most modern techniques in molecular biology: Real-time polymerase chain reaction (PCR), immunohistochemistry, and electron microscopy. The causal role for systemic active human herpesvirus (HHV)-6 and -7 infection in PR is based on the detection of their DNA in plasma, mRNA expression, and antigens in skin lesions of PR patients. Notably, HHV-6 and HHV-7 plasma viremia, marker of systemic active infection, has been demonstrated in PR and related to the presence of constitutional symptoms.[2,3] Accordingly, the cytopathic effect and syncytia formation observed in peripheral blood mononuclear cell (PBMC) cultures from PR patients, HHV-6 and -7 mRNA expression, and specific antigens found in lesional skin, are additional evidences of productive infection. Such morphological changes were not observed in the co-cultured mononuclear cells collected from patients reassessed after the acute illness, whereas the samples collected during PR recurrences showed the same changes again.[2,3] Moreover, herpesvirus virions in various stages of morphogenesis have been detected by electron microscopy in skin lesions and in the supernatant of co-cultured PBMCs from PR patients.

Secondly, although the study of Jairath *et al*. is interesting,[1] there is still inadequate evidence for the therapeutic effect of NB-UVB irradiation in PR. The likely rationale may be the suppression of the cell-mediated immune response and modification of the number and function of Langerhans cells in the skin, facilitating improvement in patient's symptoms. Only a few studies reported that NB-UVB results in a reduced severity of PR (without changing its course) whereas other authors, finding no benefit from UVB, switched therapy to antiviral agents observing resolution within 6 days.[4]

Before starting any therapy, one should remember that PR is a self-limiting disease that spontaneously resolved in about 45 days.[3] However, in cases of extensive, relapsing/persistent disease, a treatment might be prescribed to shorten the course of the disease. Considering that PR is associated with HHV-6/7 reactivation in previously infected patients, it may be justified to administer antiviral agents, as acyclovir, mainly in the above-mentioned cases. This therapy may be effective especially in the first week after PR onset when the HHV-6/7 replicative viral activity is probably very high.[5]

Finally, to date, no treatment can be recommended on the basis of evidence-based medicine and PR needs just reassurance and a little rest. However, when occurring in pregnant women, especially during the first 15 weeks of gestation, with widespread lesions and prolonged constitutional symptoms, PR may herald a possible HHV-6/7 intrauterine fetal infection with premature delivery and fetal death. In such cases, which might affect the pregnancy outcome, appropriate antiviral therapy may be considered.[5]

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Conflicts of interest

There are no conflicts of interest.

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